960-86

Implications of Alternative Classifications of Sudden Cardiac Death: A Prospective Analysis of 109 Deaths in Defibrillator Trials

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In order to explore the implications of using varied definitions of sudden cardiac death (SCD), a classification (CL) committee (3 cardiologists) prospectively evaluated 109 deaths over a period of 19 months in patients with an implantable cardioverter defibriliator (ICD). The basis for CL was the CAST approach with additional assessments of the consequences of considering autopsy and ICD interrogation information. Concordance and/or discordance between committee members was recorded. *Results:* Of the 834 patients followed for 19 months, there were 109 deaths: 17 were classified SCD, 51 non-SCD, and 40 non-cardiac. Of the deaths classified as SCD, 10/17 were unwitnessed as compared to 6/51 non-SCD and 3/40 non-cardiac deaths; $p < 0.001$. ICD detections occurred in 5/17 SCD <1 hour, 7/17 SCD <6 hours; therefore, 10/17 SCD had no ICD detection or information available. There was committee discordance in 5/17 SCD compared to 18/51 non-SCD and 16/40 non-cardiac. SCD rates as high as 3.6% (30/834) can be estimated if all SCD cases CL by ≥1 member was counted as SCD. Likewise, a SCD rate as low as 0.8% (7/834) is possible if SCD is limited to witnessed SCD ≤1 hour; (a 4-fold difference). Autopsy information was available in 29/109 deaths. In 7 cases, autopsy findings resulted in changing a "SCD" CL (5 witnessed; 2 unwitnessed) to either non-SCD or non-cardiac [ruptured abdominal (N = 2) or thoracic aortic (N = 1) aneurysm, acute MI (N = 1), cerebral infarction (N = 1), pulmonary embolism (N = 2)]. Thus, had autopsy information been unavailable or not considered, the SCD rate would have increased to 24/834 (2.9%). ICD interrogation was unavailable in 51/109 (47%), most commonly due to being buried with the patient or programmed off prior to death. *Conclusion:* A 4-fold spectrum of SCD rates is possible to report from the identical data-set. ICD interrogation has significant limitations for use in death CL, in contrast to its utility in clinical management. Autopsy results clarify cause-specific mortality in deaths that are temporally quite "sudden." Total mortality is the most objective primary end point.

960-87

Does Monthly Fourtime Manual Capacitor Reformation Prolong the Lifetime of the Implantable Cardioverter/Defibrillator Ventak PRx® 1700/1705?

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Background: In 2 patients the implantable cardioverter/defibrillator (ICD) (Ventak® PRx 1700/1705; Cardiac Pacemakers Inc., St. Paul) had to be replaced because the device fulfilled the "elective replacement time 1" (ERT1) criterion of premature battery depletion already after 601 and 756 days (d).

Study design: In 14 patients (9 with battery status "middle of life 2" (MOL2) — the last battery status level before battery depletion — and 5 with ERT1 we studied prospectively, if a fourtime (instead of one) manual capacitor reformation and shortening of the follow-up interval to 1 month (instead of 2) prolonged the battery lifetime. In addition to the battery status level the charge time for 34 J was measured. Only if the charge time exceeded 15 s after the second capacitor reformation, ICD replacement was scheduled.

Results: The replacement of ICDs with initial battery status ERT1 was delayed for 163 ± 63 d. All devices with initial battery status MOL2 except 2 reached ERT1 and had to be exchanged after 960 ± 261 d. The mean charge time decreased significantly with the second capacitor reformation (14.96 vs. 18.50 vs. 14.59 vs. 14.16 vs. 13.83 s [prior to capacitor reformation, after 1.–4. capacitor reformation]; $p \leq 0.05$).

Conclusion: Reaching battery status MOL2 the replacement of the ICD Ventak® PRx 1700/1705 can be delayed at least 5 months by monthly fourtime manual capacitor reformation.

960-88

Factors Associated with High Impedance with a Non-Thoracotomy Defibrillation Lead System

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Peak current flow across the heart determines the success of defibrillation and is inversely dependent on the impedance between the defibrillation electrodes. Factors associated with increased impedance with implantable defibrillators using non-thoracotomy lead systems have not been well described. We compared echocardiographically-derived variables in 41 patients in whom implantation of a non-thoracotomy lead system was attempted. Defibrillation threshold and lead impedance were measured at end-expiration using a monophasic shock. Patients were divided into two groups based on impedance: low (≤47 ohms, mean 39 ± 1 ohms, N = 30) and high (>47 ohms, 51 ± 1 ohms, N = 11). Left ventricular mass, wall thickness, (posterior wall + septum), left ventricular diastolic diameter (LVDD), end diastolic and end systolic volumes (EDV, ESV) and ejection fraction were determined in the standard fashion from 2-D echocardiograms.

	≤47 ohms	>47 ohms	P value
Wall Thickness, mm	2.1 ± 0.1	2.0 ± 0.1	0.6
LVDD, mm	5.6 ± 0.2	6.1 ± 0.4	0.11
Mass, g	325 ± 18	338 ± 34	0.74
EDV, ml	208 ± 11	296 ± 27	0.001
ESV, ml	137 ± 10	204 ± 22	0.004
Ejection Fraction, %	35 ± 3	28 ± 2	0.16
Lead Alone, # (%)	9 (30%)	4 (36%)	NS
Lead + Patch, # (%)	18 (60%)	5 (46%)	NS
Required Thoracotomy, # (%)	3 (10%)	2 (18%)	NS

We conclude that: (1) patients with high defibrillation lead impedance have significantly larger EDVs and ESVs; (2) incressed impedance does not predict need for a subcutaneous patch or inability to implant a non-thoracotomy lead system.

960-89

Low Current Impedance: Does It Predict Shock Impedance in Implantable Defibrillators?

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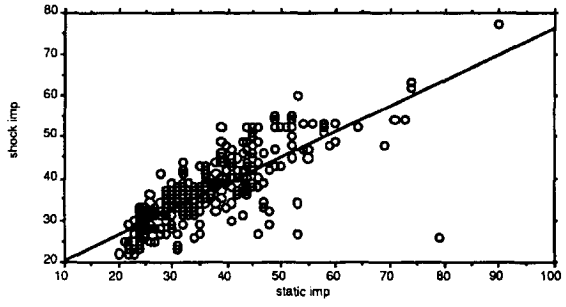
The shock impedance has become increasingly important in setting the optimal combination of pulse width and voltage for monophasic and biphasic shocks. However, many systems measure impedance using a 5 to 15J synchronized shock in sinus rhythm. We examined the ability of a low current signal static impedance (imp) (2 mA, 15 microsec) via the shocking electrodes to predict the impedance during shock delivery. The static impedance (mean 36.6 ± 11.1 ohms) was not statistically significant from the shock impedance (mean 36.9 ± 8.6 ohms). The correlation ($r = 0.81$) of the static impedance with the shock impedance in 83 patients during implantation of Telectronics 4210 implantable defibrillator is shown.



These data suggest that the low current static impedance correlates well with shock impedance and that this method may provide useful information in setting the initial combination of pulse width and voltage, obviating the need for a test shock in sinus rhythm.

960-90

Ventricular Fibrillation Induction Efficacy of Low Energy T-Wave Synchronized Shocks in Patients with Nonthoracotomy ICDs

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Incremental ventricular pacing or alternating current have been traditionally used to induce ventricular fibrillation (VF) while testing defibrillating function of the ICD. The available data are limited on ventricular fibrillation induction efficacy of the low energy T-wave shocks. To determine this, we analyzed data in 14 patients with Medtronic ICDs (7219C 9 pts, 7219D 2 pts, PCD 3 pts) who had undergone a total of 110 VF induction attempts either at time of ICD implant or during follow-up noninvasive EPS. The underlying heart disease was coronary in all patients with prior myocardial infarction in 10 patients. The LV ejection fraction was 30 ± 12% (range 15–64%), QT interval 440 ± 57 ms (range 360–560 ms) and QTc interval 449 ± 49 ms (range 336–520 ms). The low energy synchronized shocks (0.2–2J) were delivered to the T wave of last of the 8 beat paced train at 400 ms with S_1-T delays ranging between 290–340 ms.

Of 110 attempts with T wave shocks, the inducible arrhythmia was VF in 69%, nonsustained polymorphic ventricular tachycardia (VT) in 15%, monomorphic VT in 2% and none in 14%. The VF induction efficacy was 75% (69 of 92 attempts) for T-wave shock energy of >0.6J compared with 50% (9 of 18 attempts) for T-wave shock energy of ≤0.6J ($P < 0.05$). In contrast, the VF induction efficacy was unaffected by S_1-T delays ranging from

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290–330 ms. It was 80% (4/5) at S_1-T delay of 290 ms, 80% (4/5) at S_1-T delay of 300 ms, 70% (44/66) at S_1-T delay of 310 ms, 78% (7/9) at S_1-T delay of 320 ms, 74% (17/23) at S_1-T delay of 330 ms and 0% (0/2) at S_1-T delay of 340 ms. In no patients incremental ventricular pacing or alternating current had to be used to induce VF.

We conclude that a low energy (1–2J) shock, when synchronized to the paced-T wave with S_1-T delays of 290–330 ms, induces VF in over 70–80% of the attempts. This precludes the need for prolonged ventricular bursts or the use of alternating current and should, thus, be the preferred modality of choice while inducing VF to check ICD function.

960-91 Influence of T-wave Shock Energy on Ventricular Fibrillation Vulnerability in Humans

John F. Swartz, Marshall Stanton, Paul DeGroot, Rahul Mehra. *St. Francis Hospital, Tulsa, OK*

Delivery of low energy T-wave shocks may induce ventricular fibrillation (VF). Clinical use of this induction method is hindered due to the need for T-wave scanning with multiple shocks to identify the window of vulnerability (WV) to VF. The purpose of this study was to test the hypothesis that WV duration is directly related to shock strength. We studied the effect of shock strength on WV in 27 consecutive patients undergoing nonthoracotomy Medtronic 7219C/D PCD implantation or testing. WV duration was tested after a 3 pulse 400 ms drive by T-wave scanning between coupling intervals (CI) of 220–420 ms in 20 ms steps using randomized 0.6J and 2.0J truncated monophasic shocks. The results were (mean ± std):

	0.6J	2.0J	p
Min CI (ms)	270 ± 23	275 ± 28	N.S.
Max CI (ms)	312 ± 31	333 ± 30	<0.05
WV Duration (ms)	39 ± 25	56 ± 33	<0.05

The mean increase in WV duration with 2.0J shocks was 166 ± 134%, $p < 0.05$. Increased WV duration was due solely to persistence of VF inducibility further into diastole. Maximal VF induction efficacy was 20/27 (74%) patients using 2.0J at 300 ms CI. Optimal induction with 0.6J was 17/27 (63%) patients at a 280 ms CI.

We conclude that: 1) Window of vulnerability duration is energy dependent, 2) Increasing shock strength extends the vulnerable period, 3) Greatest induction efficacy for a single shock strength and CI is 74%, and 4) Need for T-wave scanning may be minimized by increasing shock energy.

961 Signal-Averaged Electrocardiography

Tuesday, March 21, 1995, Noon–2:00 p.m.
Ernest N. Morial Convention Center, Hall E
Presentation Hour: Noon–1:00 p.m.

961-76 Frequency Domain Analysis of the Signal-Averaged ECG in Patients with Arrhythmogenic Right Ventricular Dysplasia

Osamu Kinoshita, Guy Fontaine, Gilles Lascault, Joelci Tonet, Robert Frank. *Hôpital Jean Rostand, Ivry sur Seine, France*

Signal-Averaged ECG (SAECG) was recorded in 28 patients (mean 38 ± 13 years, 19 men) with arrhythmogenic right ventricular dysplasia (ARVD) who had sustained ventricular tachycardia and 35 normal subjects (mean 35 ± 11 years, 30 men). The Fast-Fourier transform analysis of SAECG was performed using a Blackman-Harris window in a segment length of 120 msec from 20 msec before the end of the QRS complex. Area ratio 1 (area of 20–50 Hz)/(area of 0–20 Hz) and area ratio 2 (area of 40–100 Hz)/(area of 0–40 Hz) were calculated. The mean value of area ratio 1 and 2 in patients with ARVD was significantly higher than those of normal subjects (410 ± 340 vs 186 ± 28; $p < 0.001$, 174 ± 143 vs 41 ± 16; $p < 0.001$). Eighteen (64%) and 21 (75%) patients had a value greater than the mean ± 2SD value of normal subjects. These results suggest that the frequency domain analysis of SAECG is an available method for detecting late potentials in patients with ARVD who have sustained ventricular tachycardia of the right ventricular origin.

961-77 The Anatomic Basis of Late Potentials in Patients with 'Idiopathic' Right Ventricular

Jaswinder S. Gill, Michael J. Davies, Edward Rowland, David E. Ward, A. John Camm. *Cardiological Sciences, St George's Hospital Medical School, London, UK*

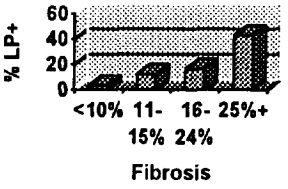
Background: A significant number of patients with 'idiopathic' right ventricular tachycardia (VT) have abnormal histology on endomyocardial biopsy and positive late potentials on signal-averaged electrocardiogram (SAE).

Aims: This study attempts to relate the histological abnormalities on endomyocardial biopsy to the presence of late potentials.

Methods: Patients with 'idiopathic' VT were studied by time domain SAE and endomyocardial biopsy. Biopsies were examined by a specialist cardiac histologist and the occurrence of abnormality related to the presence of late potentials.

Results: Fifty-two patients, mean age 34.6 ± 13.8 years were studied. Six patients had endocardial thickening, 31 had interstitial fibrosis (quantified by morphometric methods), 27 had fibrosis isolating myocardial cells into islets, 4 had leucocyte infiltration and 26 had adipose infiltration. Of these abnormalities, interstitial fibrosis was associated with the presence of late potentials on SAE (Table). There was a strong relationship between the degree of interstitial fibrosis and the prevalence of late potentials (Figure). Adipose infiltration was not related to the presence of fibrosis and not associated with the presence of late potentials on signal-averaged ECG.

	LP −/+	LP −/+	
No fibrosis	19/1	No adipose infiltr	19/6
Fibrosis	22/7	Adipose infiltr	22/2



Conclusions: The presence of increasing degrees of interstitial fibrosis relates to increasing prevalence of late potentials on signal-averaged ECG, whereas these do not appear to relate to any of the other histological abnormalities examined. This strongly suggests that interstitial fibrosis is involved in the genesis of late potentials.

961-78 Importance of Left Ventricular Ejection Fraction and Signal Averaged Electrocardiogram but not of Coronary Artery Patency nor Holter Monitoring to Predict Severe Arrhythmic Events After a First Myocardial Infarction in the Thrombolytic Era

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We followed-up 244 consecutive patients (210 men, mean age = 56 ± 9 yrs) who survived the acute phase (alive after day 7) of a first anterior (N = 102) or inferior (N = 142) myocardial infarction (MI) with a mean follow-up (FU) delay of 57 ± 18 months. In the acute phase, 97/244 patients (40%) received a thrombolytic therapy. Within the second and third week after admission, all patients underwent a Holter ECG monitoring graded by the Lown classification, a signal averaged electrocardiogram (SAECG) and a coronary angiography. Three parameters were measured by SAECG (predictor system, 40 Hz high-pass filter): total QRS duration (QRSd), root mean square voltage of the last 40 ms (RMS) and duration of the terminal low (<40 uV) amplitude signal (LAS). The number of diseased vessels as well as the infarct related artery (IRA) patency was evaluated by TIMI grading (TIMI 2 or 3 = patent) and left ventricular ejection fraction (LVEF) was measured angiographically. Cox proportional hazards model was used for the statistical analysis.

Results: We observed 18 arrhythmic events (AE): 10 sudden cardiac death and 8 ventricular tachycardia during the FU period. Statistical analysis identified 3 independent factors predictive of the occurence of an AE: 1) LVEF, with a risk multiplied by 1.9 for each 0.10 decrease in the LVEF value, 2) LAS, with a risk multiplied by 1.3 for each 5 ms increase in the LAS value and 3) absence of thrombolysis, with a risk multiplied by 3.9.

Conclusions: After MI in the thrombolytic era the Holter ECG monitoring and the results of the coronary angiography do not predict the risk of an AE. LVEF, SAECG and absence of thrombolysis are the 3 independent predictors of such a risk.

961-79 Ventricular Late Potentials and Left Ventricular Function After Early ACE-Inhibition in Myocardial Infarction

Anders Junker, Palle Ahlquist, Per Thayssen, Kristian Angelo-Nielsen, Hans Mickley, Mogens Møller. *Department of Cardiology, Odense University Hospital, Odense, Denmark*

Myocardial infarction may be followed by development of ventricular late potentials (LP) and by impairment of left ventricular function.

The purpose of the present double-blind placebo-controlled study was to